Erastin

Cat. No.: HY-15763

CAS No.: 571203-78-6

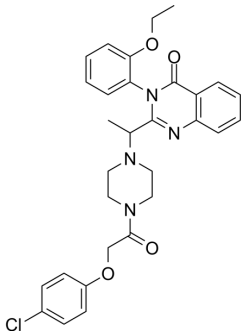
Molecular Formula: $C_{30}H_{31}ClN_4O_4$

Molecular Weight: 547.04

Target: Ferroptosis; VDAC

Pathway: Apoptosis; Membrane Transporter/Ion Channel

Storage: Powder -20°C 3 years
4°C 2 years
* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (22.85 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8280 mL	9.1401 mL	18.2802 mL
	5 mM	0.3656 mL	1.8280 mL	3.6560 mL
	10 mM	0.1828 mL	0.9140 mL	1.8280 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 5 mg/mL (9.14 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (2.29 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (1.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Erastin is a ferroptosis inducer. Erastin exhibits the mechanism of ferroptosis induction related to ROS and iron-dependent signaling. Erastin inhibits voltage-dependent anion channels (VDAC2/VDAC3) and accelerates oxidation, leading to the accumulation of endogenous reactive oxygen species. Erastin also disrupts mitochondrial permeability transition pore (mPTP) with anti-tumor activity[1][2][3].

In Vitro

Erastin (10 μM; 24 h) triggers ferroptosis in ectopic endometrial stromal cells (EESCs), and increases the total ROS level at 9 h [1].

Erastin shorts mitochondria and increases membrane density in EESCs[1].
Erastin (10 μM; 9 h) decreases the mRNA expression levels of iron-related proteins, such FPN (iron exporter) in EESCs. However, FPN overexpression significantly inhibits erastin-induced ferroptosis in EESCs[1].
Erastin (10 μM; 24 h) induces mitochondrial permeability transition pore (mPTP) opening in HT-29 colorectal cancer cells[2].
Erastin (30 μM; 72 h) significantly inhibits the growth of HT-29 colorectal cancer cells[2].
The molecular mechanism by which Erastin induces ferroptosis is related to genes regulating iron or mitochondrial fatty acid metabolism. Includes ribosomal protein L8, iron response element binding protein 2 (IREB2), ATP synthase F0 complex subunit C3, citrate synthase, tetrapeptide repeat domain 35, and acyl-CoA synthetase family member 2 (ACSF2)[3].
Note:
1. Different cell lines may have different sensitivity to a same compound. As reported, A549, HCT116, HepG2, H1299 cells may be insensitive to Erastin[3][4][5].
2. Erastin is unstable in solution. Freshly prepared is recommended.
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Normal endometrial stromal cells (NESCs) and endometrial stromal cells (EESCs)
Concentration:	0, 0.5, 0.8, 1, 1.5, 2, 2.5, 5, 10 μM
Incubation Time:	24 hours
Result:	Induced cell detachment and overt death in EESCs, but not NESCs.

Apoptosis Analysis[1]

Cell Line:	EESCs infected with adenovirus expressing FPN cDNA (co-incubation for 24 hr)
Concentration:	0, 0.5, 1.5, 2.5, 5 and 2.5 μM
Incubation Time:	24 hours
Result:	Induced ferroptosis by decreasing the levels of total ROS and lipid ROS. And reversed by the overexpression of FPN in adenovirus-infected cells.

In Vivo

Erastin can be used in animal modeling to construct ferroptosis induction model.

Erastin (40 mg/kg; i.p.; once every 3 days for 2 weeks) suppresses endometriotic implants in the mouse endometriosis model, indicating Erastin regresses ectopic lesions by trigging ferroptosis[1].
Erastin (10 mg/kg, 30 mg/kg; i.p.; once daily for 4 weeks) suppresses HT-29 xenograft growth in SCID mice, with more potent efficacy under 30 mg/kg treatment[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mouse model of endometriosis[1]
Dosage:	40 mg/kg
Administration:	Intraperitoneal injection; once every 3 days for 2 weeks
Result:	Showed little impact on body weight of mice and hair of mice displayed neat and glossy. Reduced the volume of ectopic lesions.

CUSTOMER VALIDATION

- Cell Discov. 2022 May 3;8(1):40.
- Nat Cell Biol. 2022 Feb;24(2):168-180.
- Adv Funct Mater. 2023 Apr 28.
- ACS Nano. 2023 Nov 15.
- J Clin Invest. 2024 Jan 23.

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REFERENCES

[1]. Li Y, et al. Erastin induces ferroptosis via ferroportin-mediated iron accumulation in endometriosis. Hum Reprod. 2021 Mar 18;36(4):951-964.

[2]. Xie Y, et al. Ferroptosis: process and function. Cell Death Differ. 2016 Mar;23(3):369-79.

[3]. Huo H, et al. Erastin Disrupts Mitochondrial Permeability Transition Pore (mPTP) and Induces Apoptotic Death of Colorectal Cancer Cells. PLoS One. 2016 May 12;11(5):e0154605.

[4]. Gai C, et al. MT1DP loaded by folate-modified liposomes sensitizes erastin-induced ferroptosis via regulating miR-365a-3p/NRF2 axis in non-small cell lung cancer cells. Cell Death Dis. 2020 Sep 14;11(9):751.

[5]. Yang Y, et al. Piperlongumine Inhibits Thioredoxin Reductase 1 by Targeting Selenocysteine Residues and Sensitizes Cancer Cells to Erastin. Antioxidants (Basel). 2022 Apr 4;11(4):710.

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